Preference is given to letters commenting on contributions published recently in the *JRSM*. They should not exceed 300 words and should be typed double spaced

Toxic shock syndrome and burns

The plans of Dr Davis and Dr Griffin to educate clinical staff about toxic shock syndrome (July 1996 *JRSM*, p420) are sensible. I wonder, however, if they would consider an additional measure that may minimise problems amongst children similar to the one they described (February 1996 *JRSM*, pp115P–116P). The advice given to parents of children with 'minor' burns that are not treated as inpatients can include advice specific to toxic shock syndrome[1]. It is possible to list some of the features that may be associated with TSS and to suggest that a child exhibiting any of these should be reviewed without delay.

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REFERENCE

1 Hardern R, Cavanagh S. Toxic shock syndrome in children with burns. *J A & E Med* 1995;**12**:68–9

Hydroxocobalamin versus cyanocobalamin

In calling for the withdrawal of cyanocobalamin for therapeutic use[1] I laid particular emphasis on the fact that hydroxocobalamin, and not cyanocobalamin, is a powerful cyanide antagonist.

Some patients with tobacco amblyopia fail to respond to treatment because, although hydroxocobalamin has been prescribed, cyanocobalamin has been administered in its stead. The diagnosis may then be questioned, treatment stopped and the patient condemned to a life of poor sight. Patients with tobacco amblyopia who have normal serum vitamin B_{12} levels and no evidence of defective vitamin B_{12} absorption will not need to continue with parenteral hydroxocobalamin after their visual acuity and visual fields have returned to normal, providing they abstain from further smoking.

Tobacco amblyopia is but one example of toxic amblyopia due to chronic cyanide intoxication[2]. I expressed concern that the World Health Organization's Committee on the selection of essential drugs listed only cyanocobalamin and not hydroxocobalamin, thus placing an incalculable number of patients with tobacco and tropical (nutritional) amblyopia at risk of progressive visual deterioration, as well as patients with Addisonian pernicious anaemia and other B_{12} deficiency disorders developing optic neuropathy if they are smokers.

The latest WHO report[3] has now revoked the original selection of essential drugs, in that hydroxocobalamin is included and cyanocobalamin has been omitted, which is to be welcomed.

I know of no condition in which it has been claimed that cyanocobalamin is preferable to hydroxocobalamin. Because confusion is likely to persist in the profession over the differences between various forms of vitamin B_{12} commercially available, and over their possible adverse effects in neuro-ophthalmological disorders, I would again strongly urge that it would be much safer, and no commercial disadvantage, if manufacturers withdrew cyanocobalamin in favour of hydroxocobalamin for therapeutic use.

Besides being present in tobacco-smoke and alcohol, cyanide has a world-wide distribution in the plant kingdom. Optic neuropathy, often associated with nerve deafness, myelopathy with pyramidal tract involvement and sensory ataxia, and is particularly apt to occur in tropical and subtropical countries where nutrition is poor and the indigenous population suffers from a low protein intake and high cyanide exposure from a dietary source such as unprocessed cassava roots.

What of the future? If the indiscriminate dumping of industrial toxic cyanide waste and untreated sewage continues unchecked, with the inherent risk of pollution of seas, rivers and food and water supplies, there may well come a time when more widespread chronic cyanide neurotoxicity occurs in the western hemisphere from a dietary source in persons with a genetic or acquired error of cyanide or vitamin B_{12} metabolism.

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REFERENCES

1 Freeman A G. Cyanocobalamin—a case for withdrawal: discussion paper. *J R Soc Med* 1992;**85**:686–71

2 Freeman A G. Optic neuropathy and chronic cyanide intoxication: a review. *J R Soc Med* 1988;**18**:103–6

3 World Health Organization. *The Use of Essential Drugs*: (Tech Rep Ser no. 850). Geneva: WHO. 1995:36

BSE: Cow politics revisited

The article by Dr McKee and colleagues (August 1996 *JRSM*, pp424–426) and the editorial by Mr Edney (p423) drew attention to important issues concerning the BSE affair. It should be noted, however, that mishandling by the UK government (and the Ministry of Agriculture, Food and Fisheries) of matters concerning cattle and human health applies not only to BSE[1]. The BSE/CJD saga has its historical counterpart in the story of pasteurization of cows' milk. Some 70 years ago, it was 'determined' that pasteurization effectively eliminated bovine tubercle bacilli from cows' milk, and so removed the risk of the associated non-pulmonary disease from children[2]. As for BSE[3–5] the government of the time elected to resist, consistently, a national pasteurization policy, despite being made aware of the then available scientific data, preferring to accept the erroneous counsel of their advisers to the effect that exposure to a low level of infection with live bovine mycobacteria would minimize the susceptibility of children to infection with the human tubercle bacillus. Subsequent events demonstrated the fallacy of this notion, but too late to prevent severe morbidity in some children and fatal disease in others.

Successive UK governments seem to be unable (or unwilling?) to comprehend medical scientific matters and to accept the necessity, with scientific research, of open and wide debate of a health issue that is causing concern to both the medical profession and the general public.

The pasteurization story is somewhat analogous to the current situation with BSE, but at least in the case of bovine tuberculosis the causative microbe was identified with certainty, unlike the aetiological agent of BSE which may be a prion protein[6,7].